

# POTT'S DISEASE OF THE SPINE IN CHILDREN: ITS COMPLICATIONS.\*

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THE PRINCIPAL and most important complication of Pott's disease is the tuberculous abscess, and will demand all our skill and attention. It is also the most frequent complication that may occur, although we may safely remark that, with thoroughly efficient and early treatment of the original disease, we would surely find fewer abscesses. We may add, however, that it is not an infallible evidence of incomplete or improper treatment, and one cannot always prevent the formation of an abscess. It is in fact quite remarkable that it does not occur more frequently, and produce more serious results.

When this condition occurs, we do not have the true abscess formation, as it contains no pus, primarily, and it is better to describe it under the name of "tuberculous abscess," containing tuberculous fluid, and, in addition, serum leukocytes, casein, and fibrin, more or less cheesy necrotic tissue, with frequently microscopic bone tissue, forming a sediment on standing. This fluid is sterile, unless secondary infection has occurred, when pus is then present, green or yellow, and of uniform consistency.

The abscess may be produced by certain conditions, which cannot always be determined in every case. We notice, first, the lessened power of resistance of the patient to the tuberculous invasion. This may be both general and local. There may be a history of a previous injury re-

autopsy. They are present in over 25 per cent of all cases of Pott's disease, in about 8 per cent of the cervical cases, 20 per cent of the dorsal, 72 per cent of the lumbar, and at least 50 per cent where the disease is in the lower spine. Here the vertebral bodies are larger, and offer less resistance than do those of the higher spine to the progress of the disease.

The abscess may remain stationary for a considerable time after its discovery, and should there be a rapid and notable increase in its size, we may expect that some outside infection has been received, or that rapid destructive changes are occurring. The position and size of an abscess may produce pain and discomfort, and attract in this way the attention of the patient. Pain and temperature also indicate that the abscess is nearing the surface, and always in the line of least resistance, aided by both gravity and pressure. The course of an abscess below the diaphragm is very interesting. It may enter the sheath of the psoas muscle, appearing externally upon the inner thigh as a psoas abscess. It may enter the sheath of the quadratus lumborum and appear between the twelfth rib and the crest of the ilium as a lumbar abscess. It is called an iliac abscess when it passes downward upon the iliac fascia, sometimes appearing at the outer end of Poupart's ligament. When it passes through the greater sciatic foramen, a gluteal abscess results.

The table of Treves shows the origin, location and point of appearance of these spinal tuberculous abscesses:

Variety.	Course.	Exit.
Cervical.....	(a) Anterior.....	Into the posterior wall of the pharynx.
	(b) Burrow beneath the deep fascia, into the thorax, as a mediastinal abscess.....	Into the trachea, oesophagus, or through an intercostal space.
	(c) Laterally, between longus colli and scaleni muscles.....	Posteriorly, to the sterno-mastoid.
Dorsal.....	(a) Burrow posteriorly.....	On the back or side, a short distance from the spine.
	(b) Within the psoas sheath.....	Beneath Poupart's ligament, in Scarpa's triangle.
Lumbar.....	(a) Enter psoas sheath.....	As psoas abscess.
	(b) Burrow between the fasciæ of the quadratus lumborum and abdominal muscles, through the internal oblique.....	Posteriorly beneath the external oblique, and latissimus dorsi, at the outer border of the erector spinæ muscle.
	(c) Gravitate beneath the internal iliac muscles, over the posterior brim of the pelvis, perforating the great sciatic foramen.....	As a gluteal abscess.
	(d) May be directed to the iliac region, along the aorta, and external iliac arteries.....	As a gluteal abscess.

ceived. Pyogenic infection is not required to produce the abscess, and, as above stated, many of them are sterile, or contain tubercle bacilli only. Pressure and irritation are potent factors, both in producing an abscess and increasing its size.

An abscess may escape observation during the patient's lifetime, and then be discovered at the

They may develop unilaterally or bilaterally, may appear in the mouth, thorax, in the posterior mediastinum, or passing downward therefrom, through the aortic opening in the diaphragm, form an iliac abscess. A retropharyngeal abscess may be caused by tuberculous disease in the occipito-axoid region, the fluid passing between the recti

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muscles. It may also dissect a path around the skull, or even enter the foramen magnum.

From the middle cervical region an abscess may dissect a channel between the scaleni and the longus colli, thence between the trapezius and the sterno mastoid, reaching the surface at the anterior border of the latter. In rare instances an abscess has entered the spinal canal, pleural cavity, lungs and intestines. The psoas abscess is undoubtedly of most frequent occurrence, and is seldom seen in children, except in Pott's disease, and is nearly an infallible sign of dorsal or lumbar tuberculous vertebral disease. They may open into the hip joint, giving rise to hip joint disease, or into the inguinal canal, simulating hernia.

Leukocytosis is found in some cases of abscess, although not invariably. Fever is not a constant symptom, but where a patient has had considerable fever it often proves a forerunner of an abscess, which, however, may not reach the surface for several weeks or even months. Hectic fever, together with other septic symptoms, is not usually present unless the abscess cavity has opened and infection occurred, but slight temperature is often observed without such infection.

An abscess in the lung produces less disturbance than one would expect, and the only physical signs found may be those of a chronic or acute localized pneumonia. The abscess may rupture into a bronchus and the fluid be coughed up, followed by more or less collapse, according to the size of the abscess.

A sudden discharge of pus is indicative of the rupture of an abscess into the pharynx, esophagus, intestine, or bladder. It is not unusual for an abscess to become stationary and absorption of its contents to follow.

An abscess is of serious import to the patient by causing delay in our mechanical treatment. Deformity is often increased by the abscess formation, and death may occur from asthenia and secondary surface infection. Spontaneous openings may discharge for years, unless very thoroughly treated, and with good drainage, and even then sinuses, deep and tortuous, may remain unhealed. The rupture of abscess fluid into the peritoneal cavity is usually fatal, and invariably so in case it should, in its course, communicate with a large blood vessel. As soon as recognized, the abscess fluid should be evacuated, followed by the most free and careful drainage, under perfect aseptic surroundings and precautions.

The retropharyngeal abscess may be opened from the mouth, if time is urgent, but it is better surgery to operate by external incision along the posterior border of the sterno-mastoid, in its upper third, dissecting carefully; then open and

drain thoroughly, and the opportunity for this free drainage gives this method its advantage.

An abscess of the thorax should be treated by rib resection and drainage. In those of the lower spine, treat by free incision, gently remove all possible of the abscess membrane with a small gauze sponge and pressure, flush well with salt solution and secure free drainage, with counter openings, if required. The patient should live in the open air and sunshine, and receive most careful mechanical treatment, as indicated.

It is possible in some cases to aspirate these abscesses, repeating as often as the cavity re-fills, and following with a pressure compress. Where the abscess is still uninfected, there is good authority for the treatment by injection of sterilized emulsion of iodoform and glycerin, in 10 to 20 per cent, using 3jj to 3iv, once in one to two weeks, either with or without previous evacuation of the contents. The toleration of the patient for iodoform should be watched, and with this in mind, and with aseptic precautions, no harm has resulted. Iodoform has no value where the abscess cavity has become infected, nor does it have much effect upon the general tuberculous process. A favorable result may be expected where the abscess contents are transformed into a viscid fluid, and from one to six injections are usually sufficient to produce this effect in susceptible cases.

While not so frequently occurring as abscess, as a complication in Pott's disease, paralysis is seen in from 5½ to 13.7 per cent of all cases, according to different observers. It is not as serious a complication, however, and, like abscess, may and does occur in some cases where early and approved methods of treatment are employed. The principal cause of paralysis is pressure, from some source, somewhere upon the cord or nerves. There may be swelling, and hypertrophy of the softer tissues within the bony canal, thickening of the dura, and pachymeningitis resulting from extension of the tuberculous process backward through the posterior ligaments, and invasion of the epidural space. There may be pressure from a necrosed fragment, or from a neighboring abscess.

Paralysis develops more often in cases where kyphosis is moderate or slight, and where the disease is located in the upper and middle dorsal region, as here the spinal canal is smaller, and it is more difficult to secure and maintain proper and efficient fixation treatment. It is said that 15 per cent of all these cases may show more or less paralysis symptoms during the progress of cure. An early symptom of paralysis is an attack of herpes zoster along the course of the nerve first sustaining pressure. Paralysis has been observed before the deformity was apparent,

but it usually develops from six months to one and a half years after the tuberculous process has begun.

The shambling gait and manner in which the patient will drag his leg, will usually first attract attention, and there will also be noticed undue muscular fatigue and weariness on slight exertion, and there soon appears the inability to stand erect. The exaggerated reflexes are shown upon examination of patella and ankle, and still later on, muscular rigidity develops. Motor paralysis precedes the sensory, and sensation may remain after loss of motion, as the anterolateral columns receive the most pressure, but if the case continues to progress unfavorably, there will be more or less sensory paralysis.

It is very necessary to distinguish true paralysis from cases that may simulate this condition, in those patients who are simply bedridden, but are not paralyzed, and whose reflexes are normal. They may be asthenic, or have contraction of the psoas muscle and may be discouraged and believe that they cannot walk. Both legs of a patient may become equally affected at the same time, or one may precede, and the same may be noticed in convalescence.

In the early stage, where weakness is about the only symptom, spastic rigidity of the limbs may be produced by moving them, or by stimulating the reflexes. Sensation, in the mild cases, although not entirely lost, is nevertheless impaired, and so, also, is control of the bladder, while that of the sphincter ani is but slightly affected. Pain is rare in paralysis, and, if present, is but the usual characteristic pain of the disease. The extent of the paralysis naturally depends greatly upon the location of the cord pressure.

In convalescence the first favorable indication is lessening of the rigidity, with slight return of motion to the toes and feet, with more or less rapid improvement thereafter. The exaggerated reflexes may persist for quite a long period after recovery, and many patients show them who were not paralyzed.

Prognosis is, on the whole, fairly good in these cases of paralysis, and recovery may safely be anticipated in 70 per cent, and may occur early if proper treatment has been instituted. It is usually complete without subsequent traces. The prognosis depends greatly, of course, upon the extent and amount of cord pressure, as shown by the degree of paralysis, and recovery will be slower and more doubtful in those cases where the sphincters are much involved, but may eventually occur even in the worst and apparently most hopeless cases.

There may be a recurrence of the symptoms after an apparent recovery, and this has been reported from one to seven times. These relapses may be possible where treatment has been either neglected altogether, or immobilization of the spine has been too brief.

The treatment of paralysis is mechanical, and thoroughly efficient treatment of the disease in general fixation of the spine as perfectly as possible. This needs particular emphasis, as there is sometimes an effort made to have the physician relax the immobilization treatment at this very time when greatest care and vigilance are imperatively indicated. During convalescence, massage and manipulation of the limbs should be faithfully employed, but these methods are contra-indicated in the early period, being then liable to overstimulate the nerve centers.

Iodid of potassium may be administered in small doses, gradually increasing to a considerable dosage, then slowly decreasing to the original amount. The operation of laminectomy is not now recommended except in rare and selected cases, and is seldom indicated.

#### THE TREATMENT OF SCLEROSIS OF THE MIDDLE EAR.\*

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BY SCLEROSIS of the middle ear I mean that condition in which the mucous membrane lining the cavity has become highly atrophic, the ossicles rigid, and the bone-conduction, in many cases, somewhat lowered from disuse. I think it worth while to define the term, as many writers use it rather loosely, including those conditions which are really the initiatory stages of sclerosis, such as hypertrophic otitis media chronica.

The many methods proposed for treating sclerosis of the middle ear, most of which were brought out with a great flourish of trumpets, only to sink into oblivion soon after, prove that we are yet a great way from having found a really good method of relieving this most distressing condition. The great activity in this direction, however, promises that before long we may arrive at some method which will be of permanent value.

Not so many years ago the sufferers with sclerosis were refused all treatment by honest aurists, and were advised to resign themselves to their condition. To-day, however, those in whom the bone-conduction is still good can have the satisfaction of trying some of the methods which have proven themselves efficacious in similar cases. Where the bone, or tissue-perception, is

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